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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	Jan 25	BLAST(R) searching in REGISTRY available in STN on the Web
NEWS 3	Jan 29	FSTA has been reloaded and moves to weekly updates
NEWS 4	Feb 01	DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS 5	Feb 19	Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 6	Mar 08	Gene Names now available in BIOSIS
NEWS 7	Mar 22	TOXLIT no longer available
NEWS 8	Mar 22	TRCTHERMO no longer available
NEWS 9	Mar 28	US Provisional Priorities searched with P in CA/CAplus and USPATFULL
NEWS 10	Mar 28	LIPINSKI/CALC added for property searching in REGISTRY
NEWS 11	Apr 02	PAPERCHEM no longer available on STN. Use PAPERCHEM2 instead.
NEWS 12	Apr 08	"Ask CAS" for self-help around the clock
NEWS 13	Apr 09	BEILSTEIN: Reload and Implementation of a New Subject Area
NEWS 14	Apr 09	ZDB will be removed from STN
NEWS 15	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS 16	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 17	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 18	Apr 22	Federal Research in Progress (FEDRIP) now available

NEWS EXPRESS	February 1 CURRENT WINDOWS VERSION IS V6.0d, CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP), AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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NEWS LOGIN	Welcome Banner and News Items
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FILE 'HOME' ENTERED AT 18:12:40 ON 23 MAY 2002

=> file medline, uspatful, dgene, embase, biosis

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 18:12:57 ON 23 MAY 2002

FILE 'USPATFULL' ENTERED AT 18:12:57 ON 23 MAY 2002
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FILE 'DGENE' ENTERED AT 18:12:57 ON 23 MAY 2002
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FILE 'BIOSIS' ENTERED AT 18:12:57 ON 23 MAY 2002
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=> s XRCC4 and DNA ligase IV

L1 134 XRCC4 AND DNA LIGASE IV

=> s l1 and binding

L2 67 L1 AND BINDING

=> s l2 and inhibition

L3 3 L2 AND INHIBITION

=> d l3 ti abs ibib tot

L3 ANSWER 1 OF 3 MEDLINE

TI The catalytic subunit DNA-dependent protein kinase (DNA-PKcs) facilitates recovery from radiation-induced **inhibition** of DNA replication.

AB Exposure of cells to ionizing radiation inhibits DNA replication in a dose-dependent manner. The dose response is biphasic and the initial steep component reflects **inhibition** of replicon initiation thought to be mediated by activation of the S-phase checkpoint. In mammalian cells, **inhibition** of replicon initiation requires the ataxia telagiectasia mutated (ATM) gene, a member of the phosphatidyl inositol kinase-like (PIKL) family of protein kinases. We studied the effect on replicon initiation of another member of the PI-3 family of protein kinases, the catalytic subunit of DNA-dependent protein kinase (DNA-PKcs) by measuring either total DNA synthesis, or size distribution of nascent DNA using alkaline sucrose gradient centrifugation. Exposure of human cells proficient in DNA-PKcs (HeLa or M059-K) to 10 Gy inhibited replicon initiation in a time-dependent manner. **Inhibition** was at a maximum 1 h after irradiation and recovered at later times. Similar treatment of human cells deficient in DNA-PKcs (M059-J) inhibited replicon initiation to a similar level and with similar kinetics; however, no evidence for recovery, or only limited recovery, was observed for up to 8 h after irradiation. In addition a defect was observed in the maturation of nascent DNA. Similarly, a Chinese hamster cell line deficient in

DNA-PKcs (irs-20) showed little evidence for recovery of DNA replication **inhibition** up to 6 h after irradiation, whereas the parental CHO cells showed significant recovery and an irs-20 derivative expressing the human DNA-PKcs complete recovery within 4 h. Normal kinetics of recovery were observed in xrs-5 cells, deficient in Ku80; in 180BR cells, deficient in **DNA ligase IV**; as well as XR-1 cells, deficient in **XRCC4**, an accessory factor of **DNA ligase IV**. Since all these cell lines share the DNA double strand break rejoining defect of M059-J and irs20 cells, the lack of recovery of DNA replication in the latter cells may not be attributed entirely to the prolonged presence of unrepaired DNA dsb. We propose that DNA-PKcs, in addition to its functions in the rejoining of DNA dsb and in DNA replication, also operates in a pathway that in normal cells facilitates recovery of DNA replication after irradiation.

ACCESSION NUMBER: 2000133036 MEDLINE
DOCUMENT NUMBER: 20133036 PubMed ID: 10666461
TITLE: The catalytic subunit DNA-dependent protein kinase (DNA-PKcs) facilitates recovery from radiation-induced **inhibition** of DNA replication.
AUTHOR: Guan J; DiBiase S; Iliakis G
CORPORATE SOURCE: Department of Radiation Oncology of Kimmel Cancer Center, Thompson Building, Jefferson Medical College, Philadelphia, PA 19107, USA.
CONTRACT NUMBER: CA 42026 (NCI)
CA 56706 (NCI)
P30 CA56036=03 (NCI)
SOURCE: NUCLEIC ACIDS RESEARCH, (2000 Mar 1) 28 (5) 1183-92. Journal code: 0411011. ISSN: 1362-4962.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; S
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000505
Last Updated on STN: 20020420
Entered Medline: 20000424

had date

L3 ANSWER 2 OF 3 USPATFULL
TI Human stress array
AB Human stress arrays and methods for their use are provided. The subject arrays include a plurality of polynucleotide spots, each of which is made up of a polynucleotide probe composition of unique polynucleotides corresponding to a human stress gene. The subject arrays find use in hybridization assays, particularly in assays for the identification of differential gene expression of human stress genes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:16850 USPATFULL
TITLE: Human stress array
INVENTOR(S): Chenchik, Alex, Palo Alto, CA, UNITED STATES
Lukashev, Matvey E., Newton, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002009730	A1	20020124
APPLICATION INFO.:	US 2001-782909	A1	20010213 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1999-441920, filed on 17 Nov 1999, UNKNOWN
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Bret E. Field, BOZICEVIC, FIELD & FRANCIS LLP, 200 Middlefield Road, Suite 200, Menlo Park, CA, 94025

NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
LINE COUNT: 877
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L3 ANSWER 3 OF 3 USPATFULL
TI Inhibitors of alternative alleles of genes encoding products that mediate cell response to environmental changes
AB Disclosed are methods for the treatment of proliferative disorders using compounds and/or environmental conditions which result in a difference in sensitivity of targeted and non-targeted cells. Certain of the methods involve the identification and use of allele-specific inhibitors of conditionally essential genes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

ACCESSION NUMBER:	2001:36603 USPATFULL		
TITLE:	Inhibitors of alternative alleles of genes encoding products that mediate cell response to environmental changes		
INVENTOR(S):	Housman, David E., Newton, MA, United States Ledley, Fred D., Needham, MA, United States Stanton, Jr., Vincent P., Belmont, MA, United States		
PATENT ASSIGNEE(S):	Variagenics, Inc., Cambridge, MA, United States (U.S. corporation)		
	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6200754	B1	20010313
APPLICATION INFO.:	US 1998-45054		19980319 (9)
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Schwartzman, Robert A.		
ASSISTANT EXAMINER:	Epps, Janet L.		
NUMBER OF CLAIMS:	13		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	3 Drawing Figure(s); 3 Drawing Page(s)		
LINE COUNT:	3654		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d his

(FILE 'HOME' ENTERED AT 18:12:40 ON 23 MAY 2002)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, BIOSIS' ENTERED AT 18:12:57 ON 23 MAY 2002
L1 134 S XRCC4 AND DNA LIGASE IV
L2 67 S L1 AND BINDING
L3 3 S L2 AND INHIBITION

=> s XRCC4

L4 252 XRCC4

=> s DNA ligase IV

L5 226 DNA LIGASE IV

=> s l4 and l5

L6 134 L4 AND L5

=> s l6 and binding interaction

L7 1 L6 AND BINDING INTERACTION

=> d l7 ti abs ibib tot

L7 ANSWER 1 OF 1 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI Functional characterization of cysteine mutants of the double-strand break repair and V(D)J recombination protein **XRCC4**.
AB **XRCC4** is a ubiquitously expressed molecule which functions in both DNA repair and V(D)J recombination. Cloning of this molecule in 1995 provided no clue to its biologic function(s), since it showed no homology with any known molecules, though more recent work has established that at least part of **XRCC4**'s role in DNA repair and V(D)J recombination is as a co-factor for **DNA ligase IV**. Previous studies have shown all the functions of **XRCC4** to be colocalized to a region encompassing amino acids 18-204, but no results have been reported which attribute functions of **XRCC4** to particular residues. We have been interested in examining the importance of cysteine residues on various aspects of **XRCC4** function. We had observed previously that iodoacetamide treatment agrogated **XRCC4** enhancement of **DNA ligase IV** activity. This suggested that one or more reactive cysteines might be involved in **XRCC4** function. To address this issue, we have generated site specific mutants to analyze the importance of the four conserved cysteines in **XRCC4**. Mutant molecules were analyzed for their ability to support V(D)J recombination, normal radioresistance, DNA **binding, interaction** with **DNA ligase IV**, and stimulation of ligase IV activity. Our analysis has shown that certain cysteine mutants exhibit a partial phenotype with respect to DNA repair and V(D)J recombination. These findings are similar to recent reports in the literature regarding partial pheotypes for other participants in these pathways. Our data provides the first evidence of the importance of particular residues in aspects of **XRCC4** function.
ACCESSION NUMBER: 2001:265920 BIOSIS
DOCUMENT NUMBER: PREV200100265920
TITLE: Functional characterization of cysteine mutants of the double-strand break repair and V(D)J recombination protein **XRCC4**.
AUTHOR(S): Streater, Gail Ferrell (1); Meek, Katheryn D.
CORPORATE SOURCE: (1) UTSWMC, 5323 Harry Hines Blvd., Dallas, TX, 75234-8884 USA
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A700. print.
Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001
ISSN: 0892-6638.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

bad date

=> d his

(FILE 'HOME' ENTERED AT 18:12:40 ON 23 MAY 2002)

FILE 'MEDLINE, USPATFULL, DGENE, EMBASE, BIOSIS' ENTERED AT 18:12:57 ON

23 MAY 2002
L1 134 S XRCC4 AND DNA LIGASE IV
L2 67 S L1 AND BINDING
L3 3 S L2 AND INHIBITION
L4 252 S XRCC4
L5 226 S DNA LIGASE IV
L6 134 S L4 AND L5
L7 1 S L6 AND BINDING INTERACTION

=> s l6 and inhibition

L8 6 L6 AND INHIBITION

=> d l8 ti abs ibib tot

L8 ANSWER 1 OF 6 MEDLINE
TI The catalytic subunit DNA-dependent protein kinase (DNA-PKcs) facilitates recovery from radiation-induced **inhibition** of DNA replication.
AB Exposure of cells to ionizing radiation inhibits DNA replication in a dose-dependent manner. The dose response is biphasic and the initial steep component reflects **inhibition** of replicon initiation thought to be mediated by activation of the S-phase checkpoint. In mammalian cells, **inhibition** of replicon initiation requires the ataxia telagiectasia mutated (ATM) gene, a member of the phosphatidyl inositol kinase-like (PIKL) family of protein kinases. We studied the effect on replicon initiation of another member of the PI-3 family of protein kinases, the catalytic subunit of DNA-dependent protein kinase (DNA-PKcs) by measuring either total DNA synthesis, or size distribution of nascent DNA using alkaline sucrose gradient centrifugation. Exposure of human cells proficient in DNA-PKcs (HeLa or M059-K) to 10 Gy inhibited replicon initiation in a time-dependent manner. **Inhibition** was at a maximum 1 h after irradiation and recovered at later times. Similar treatment of human cells deficient in DNA-PKcs (M059-J) inhibited replicon initiation to a similar level and with similar kinetics; however, no evidence for recovery, or only limited recovery, was observed for up to 8 h after irradiation. In addition a defect was observed in the maturation of nascent DNA. Similarly, a Chinese hamster cell line deficient in DNA-PKcs (irs-20) showed little evidence for recovery of DNA replication **inhibition** up to 6 h after irradiation, whereas the parental CHO cells showed significant recovery and an irs-20 derivative expressing the human DNA-PKcs complete recovery within 4 h. Normal kinetics of recovery were observed in xrs-5 cells, deficient in Ku80; in 180BR cells, deficient in **DNA ligase IV**; as well as XR-1 cells, deficient in **XRCC4**, an accessory factor of **DNA ligase IV**. Since all these cell lines share the DNA double strand break rejoining defect of M059-J and irs20 cells, the lack of recovery of DNA replication in the latter cells may not be attributed entirely to the prolonged presence of unrepaired DNA dsb. We propose that DNA-PKcs, in addition to its functions in the rejoining of DNA dsb and in DNA replication, also operates in a pathway that in normal cells facilitates recovery of DNA replication after irradiation.
ACCESSION NUMBER: 2000133036 MEDLINE
DOCUMENT NUMBER: 20133036 PubMed ID: 10666461
TITLE: The catalytic subunit DNA-dependent protein kinase (DNA-PKcs) facilitates recovery from radiation-induced **inhibition** of DNA replication.
AUTHOR: Guan J; DiBiase S; Iliakis G
CORPORATE SOURCE: Department of Radiation Oncology of Kimmel Cancer Center, Thompson Building, Jefferson Medical College, Philadelphia,

PA 19107, USA.
CONTRACT NUMBER: CA 42026 (NCI)
CA [redacted]706 (NCI)
P30 CA56036=03 (NCI)
SOURCE: NUCLEIC ACIDS RESEARCH, (2000 Mar 1) 28 (5) 1183-92.
Journal code: 0411011. ISSN: 1362-4962.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; S
ENTRY MONTH: 200004
ENTRY DATE: Entered STN: 20000505
Last Updated on STN: 20020420
Entered Medline: 20000424

L8 ANSWER 2 OF 6 USPATFULL
TI Human stress array
AB Human stress arrays and methods for their use are provided. The subject arrays include a plurality of polynucleotide spots, each of which is made up of a polynucleotide probe composition of unique polynucleotides corresponding to a human stress gene. The subject arrays find use in hybridization assays, particularly in assays for the identification of differential gene expression of human stress genes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2002:16850 USPATFULL
TITLE: Human stress array
INVENTOR(S): Chenchik, Alex, Palo Alto, CA, UNITED STATES
Lukashev, Matvey E., Newton, MA, UNITED STATES

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 2002009730	A1	20020124
APPLICATION INFO.:	US 2001-782909	A1	20010213 (9)

RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1999-441920, filed on 17 Nov 1999, UNKNOWN
DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: Bret E. Field, BOZICEVIC, FIELD & FRANCIS LLP, 200 Middlefield Road, Suite 200, Menlo Park, CA, 94025
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
LINE COUNT: 2377
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 3 OF 6 USPATFULL
TI Inhibitors of alternative alleles of genes encoding products that mediate cell response to environmental changes
AB Disclosed are methods for the treatment of proliferative disorders using compounds and/or environmental conditions which result in a difference in sensitivity of targeted and non-targeted cells. Certain of the methods involve the identification and use of allele-specific inhibitors of conditionally essential genes.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
ACCESSION NUMBER: 2001:36603 USPATFULL
TITLE: Inhibitors of alternative alleles of genes encoding products that mediate cell response to environmental changes
INVENTOR(S): Housman, David E., Newton, MA, United States
Ledley, Fred D., Needham, MA, United States
Stanton, Jr., Vincent P., Belmont, MA, United States

PATENT ASSIGNEE(S): Variagenics, Inc., Cambridge, MA, United States (U.S. Corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6200754	B1	20010313
APPLICATION INFO.:	US 1998-45054		19980319 (9)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Schwartzman, Robert A.
ASSISTANT EXAMINER: Epps, Janet L.
NUMBER OF CLAIMS: 13
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT: 3654
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L8 ANSWER 4 OF 6 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

TI The catalytic subunit DNA-dependent protein kinase (DNA-PKcs) facilitates recovery from radiation-induced **inhibition** of DNA replication.

AB Exposure of cells to ionizing radiation inhibits DNA replication in a dose-dependent manner. The dose response is biphasic and the initial steep component reflects **inhibition** of replicon initiation thought to be mediated by activation of the S-phase checkpoint. In mammalian cells, **inhibition** of replicon initiation requires the ataxia telagiectasia mutated (ATM) gene, a member of the phosphatidyl inositol kinase-like (PIKL) family of protein kinases. We studied the effect on replicon initiation of another member of the PI-3 family of protein kinases, the catalytic subunit of DNA-dependent protein kinase (DNA-PKcs) by measuring either total DNA synthesis, or size distribution of nascent DNA using alkaline sucrose gradient centrifugation. Exposure of human cells proficient in DNA-PKcs (HeLa or M059-K) to 10 Gy inhibited replicon initiation in a time-dependent manner. **Inhibition** was at a maximum 1 h after irradiation and recovered at later times. Similar treatment of human cells deficient in DNA-PKcs (M059-J) inhibited replicon initiation to a similar level and with similar kinetics; however, no evidence for recovery, or only limited recovery, was observed for up to 8 h after irradiation. In addition a defect was observed in the maturation of nascent DNA. Similarly, a Chinese hamster cell line deficient in DNA-PKcs (irs-20) showed little evidence for recovery of DNA replication **inhibition** up to 6 h after irradiation, whereas the parental CHO cells showed significant recovery and an irs-20 derivative expressing the human DNA-PKcs complete recovery within 4 h. Normal kinetics of recovery were observed in xrs-5 cells, deficient in Ku80; in 180BR cells, deficient in **DNA ligase IV**; as well as XR-1 cells, deficient in **XRCC4**, an accessory factor of **DNA ligase IV**. Since all these cell lines share the DNA double strand break rejoining defect of M059-J and irs20 cells, the lack of recovery of DNA replication in the latter cells may not be attributed entirely to the prolonged presence of unrepaired DNA dsb. We propose that DNA-PKcs, in addition to its functions in the rejoining of DNA dsb and in DNA replication, also operates in a pathway that in normal cells facilitates recovery of DNA replication after irradiation.

ACCESSION NUMBER: 2000072591 EMBASE
TITLE: The catalytic subunit DNA-dependent protein kinase (DNA-PKcs) facilitates recovery from radiation-induced **inhibition** of DNA replication.
AUTHOR: Guan J.; DiBiase S.; Iliakis G.
CORPORATE SOURCE: G. Iliakis, Department of Radiation Oncology, Kimmel Cancer Center, Jefferson Medical College, Thompson Building,

Philadelphia, PA 19107, United States.
ge[redacted]e.iliakis@mail.tju.edu
SOURCE: Nu[redacted]ic Acids Research, (1 Mar 2000) [redacted]8/5 (1183-1192).
Refs: 73
ISSN: 0305-1048 CODEN: NARHAD
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 022 Human Genetics
029 Clinical Biochemistry
LANGUAGE: English
SUMMARY LANGUAGE: English

L8 ANSWER 5 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI The catalytic subunit of DNA-dependent protein kinase (DNA-PKcs) facilitates recovery from radiation-induced **inhibition** of DNA replication.
ACCESSION NUMBER: 2001:440389 BIOSIS
DOCUMENT NUMBER: PREV200100440389
TITLE: The catalytic subunit of DNA-dependent protein kinase (DNA-PKcs) facilitates recovery from radiation-induced **inhibition** of DNA replication.
AUTHOR(S): Guan, Jun (1); DiBiase, Steven J.; Iliakis, George
CORPORATE SOURCE: (1) Department of Radiation Oncology, University of Maryland Medical Center, Baltimore, MD USA
SOURCE: Proceedings of the American Association for Cancer Research
Annual Meeting, (March, 2001) Vol. 42, pp. 413. print.
Meeting Info.: 92nd Annual Meeting of the American Association for Cancer Research New Orleans, LA, USA March 24-28, 2001
ISSN: 0197-016X.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

L8 ANSWER 6 OF 6 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
TI The catalytic subunit DNA-dependent protein kinase (DNA-PKcs) facilitates recovery from radiation-induced **inhibition** of DNA replication.
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in **DNA ligase IV**; as well as XR-1 cells, deficient in **XRCC4**, an accessory factor of **DNA ligase IV**. Since all these cell lines share the DN double strand break rejoining defect of M059-J and irs20 cells, the lack of recovery of DNA replication in the latter cells may not be attributed entirely to the prolonged presence of unrepaired DNA dsb. We propose that DNA-PKcs, in addition to its functions in the rejoining of DNA dsb and in DNA replication, also operates in a pathway that in normal cells facilitates recovery of DNA replication after irradiation.

ACCESSION NUMBER: 2000:233348 BIOSIS
DOCUMENT NUMBER: PREV200000233348
TITLE: The catalytic subunit DNA-dependent protein kinase (DNA-PKcs) facilitates recovery from radiation-induced **inhibition** of DNA replication.
AUTHOR(S): Guan, Jun; DiBiase, Steven; Iliakis, George (1)
CORPORATE SOURCE: (1) Department of Radiation Oncology of Kimmel Cancer Center, Jefferson Medical College, Thompson Building, Philadelphia, PA, 19107 USA
SOURCE: Nucleic Acids Research, (March 1, 2000) Vol. 28, No. 5, pp.
1183-1192.
ISSN: 0305-1048.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English